those not so treated. In an attempt to demonstrate this more objectively, patients on the second day of the puerperium were asked to signify by a "Yes" or a "No" whether their perineums were (i) free from discomfort, (ii) uncomfortable, (iii) painful, (iv) very painful. The results are shown in the Table.

We intend to try this method of treatment immediately post-operatively with patients who have had other forms of perineal surgery.

We are indebted to Mr. Tomkinson, who allowed us to use his patients as controls, and to Sisters Farebrother, Langridge, and Lewis for their enthusiastic help. The plastic packs have been made for us by John Tye and Son Ltd., 457 Caledonian Road, N.7.

—We are, etc.,

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Aplastic Anaemia due to Potassium Perchlorate

SIR,—Having read with interest the reports of Dr. Q. J. G. Hobson (May 13, p. 1368) and Drs. R. Sleigh Johnson and W. G. Moore (p. 1369), we wish to record a similar case of aplastic anaemia due to potassium perchlorate. This differs from these two cases in that a dose of 600 mg. per day was never exceeded; also no other drug or dose of radioiodine was administered, so that the cause would appear definitely to be due to the use of potassium perchlorate.

A 54-year-old woman was thought to be mildly thyrotoxic in December, 1959, following a dental extraction: in March, 1960, she had lost a further 5 lb. (2.3 kg.) in weight and was started on potassium perchlorate, 200 mg. three times daily, which was reduced in August, 1960, to 200 mg. twice daily. In October she re-attended with a four weeks' history of bruising and purpura; the drug was then withdrawn. Blood count showed: haemoglobin 40%, R.B.C.s 2,000,000, W.B.C.s 2,000 per c.mm. (14% polymorphs, 86% lymphocytes), platelets 100,000 per c.mm., reticulocytes 0.5%. Bone-marrow examinations in October, November, and December, 1960, showed almost complete absence of red- and white-cell precursors. Despite treatment with prednisolone, 60 mg., and testosterone, 40 mg., daily, and fresh blood transfusions and antibiotics, she died in January, 1961, with extensive purpura and high fever. Post-mortem examination showed haemorrhages in all organs and complete marrow aplasia.

In this case marrow aplasia occurred only after six months' treatment with potassium perchlorate, 600 mg. and later 400 mg., daily, and, despite survival for three months with transfusion, steroids, and androgens, the bone marrow showed no sign of recovery. Our experience also confirms that patients need to be warned to stop the drug if any unusual rash or other symptoms appear, as our patient continued with the tablets for four weeks after the appearance of purpura. Dr. Hobson's suggestion of a printed warning on the container of such tablets might well prevent a recurrence of this disaster.—We are, etc.,

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Fat-malabsorption in Congestive Heart Failure

SIR,—In his article on fat-malabsorption in congestive heart failure (May 6, p. 1276), Dr. R. Vaughan Jones mentions pancreatic malfunction as a possible factor in the pathogenesis of this disorder. His results substantiate those of Hakkila *et al.*,[1] who found evidence of diminished lipid-bound activity in the blood and of greatly increased faecal-fat activity in cases of cardiac failure as compared with a control group. The latter authors also consider the possibility of deficient enzyme-secretion as one of several factors concerned, but in neither article is there reference to a specific pancreatic lesion. That such a lesion does exist, however, can be confirmed by examining sections of the pancreas in those cases of cardiac failure which come to necropsy.

In reviewing a large series of routine necropsies performed at Manchester Royal Infirmary, I have been impressed by the occurrence of a lesion of the pancreatic exocrine-tissue, confined exclusively to cases dying in congestive cardiac failure, which I have termed "peripheral acinar atrophy." Briefly, the lesion consists of zones of atrophic acini situated at the periphery of the primary pancreatic lobule, associated with distension of the interacinar capillary plexus and oedema of the interstitial tissue; the acini at the centre of the lobule and the island of Langerhans are of normal appearance. The peripheral distribution of these atrophic zones is of interest, being governed apparently by the peculiar vascular supply of the lobule in which the entering arterioles, while giving one or more definite arteriolar branches to the island, form a diffuse inter-acinar capillary plexus. The venous effluent is collected from the periphery of the lobule and drains into the veins of the interlobular connective tissue. It would appear, therefore, that the island and its adjacent acini are in immediate relationship to the arterial side of the capillary network but that peripherally situated acini are related more to the venous side of the inter-acinar capillary plexus. Thus in congestive cardiac failure, when tissue nutrition is imperilled by rising venous pressure combining with circulatory insufficiency to produce deficient oxygenation, the periphery of the lobule is more liable to undergo anoxic degenerative change.

All the patients in whom the lesion occurred were in terminal congestive cardiac failure resulting from a variety of cardiovascular and pulmonary conditions. Both the incidence and the severity of the lesion in the pancreas were directly proportional to the duration of the terminal episode of cardiac failure. The analogy of this lesion with that seen in the liver in passive venous congestion will be immediately obvious, and therefore it might be termed more generally "chronic venous congestion of the pancreas," though this does not denote its peculiar territorial distribution. My observations confirm those of von Glahn and Chobot,[2] who also noted pancreatic atrophy in cardiac failure. These authors apart, I have been unable to find any reference to the lesion or its effect on pancreatic function, though most histopathologists must be aware of its existence, if not of its significance, which has now been emphasized by Dr. Vaughan Jones and other workers in this field.

Whether the lesion indicates pancreatic dysfunction could not be determined in my retrospective study, as serum-enzyme estimations were not available. However, Gray *et al.*[3] have reported subnormal levels of blood

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